**U.S. Department of Labor**

Office of Administrative Law Judges
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**Issue Date: 03 August 2004**

Case Nos. 2003-BLA-80
2003-BLA-5299

In the Matter of

THOMAS A. JONES (Deceased), AND
FRANCES JONES, Survivor of THOMAS A. JONES,
Claimant,

v.

ELKAY MINING COMPANY,
Employer,

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS,
Party-in-Interest.

Appearances: S.F. Raymond Smith, Esq.
For the Claimant

Ashley M. Harman, Esq.
For the Employer

Before: MICHAEL P. LESNIAK
Administrative Law Judge

**DECISION AND ORDER — DENYING BENEFITS IN MINER'S AND SURVIVOR'S CLAIMS**

This case arises from a claim for benefits under the "Black Lung Benefits Act," Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. § 901 *et seq*. (hereinafter referred to as "the Act"), and applicable federal regulations, mainly 20 C.F.R. Parts 412, 718, and 725 ("Regulations").

Benefits under the Act are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis or to the survivors of persons whose death was

caused by pneumoconiosis. Coal Workers' Pneumoconiosis (CWP) is a dust disease of the lung arising from coal mine employment and is commonly known as black lung.[1]

A formal hearing was conducted in Charleston, West Virginia on August 27, 2003, at which all parties were afforded a full opportunity to present evidence and argument, as provided in the Act and Regulations issued thereunder.[2] The parties timely filed a joint "Stipulation of Evidence" on August 11, 2003. Employer filed its closing comments on December 2, 2003.

## ISSUES

The contested issues are:

1) Whether the miner had pneumoconiosis;
2) Whether the pneumoconiosis arose out of the miner's coal mine employment;
3) Whether the miner was totally disabled due to pneumoconiosis;
4) Whether the miner has established a change in condition pursuant to 20 C.F.R. §§ 725.309 and 725.310 (2000) (duplicate claim and modification);
5) Whether there was a mistake in a determination of fact (modification); and
6) Whether the miner's death was due to pneumoconiosis.

## FINDINGS OF FACT AND CONCLUSIONS OF LAW

### Procedural History and Factual Background [3]

#### Procedural History

The miner, Thomas A. Jones, was born on July 2, 1923 and married his wife, Frances, on June 13, 1986. DX 12. The miner's first claim for benefits, filed in 1970 with the Social Security Administration, was finally denied. DX 28. The miner filed his second claim on February 9, 1987; it was denied by the District Director on May 13, 1987. The miner did not appeal the denial. DX 29.[4] The miner filed his third claim on April 9, 1998; the District Director awarded benefits. Employer appealed, the claim was subsequently denied by Administrative Law Judge Leland on November 30, 1999, and the denial was affirmed by the Benefits Review

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[1] The following abbreviations have been used in this decision: DX = Director's exhibit; EX = Employer's exhibit; CX = Claimant's exhibit; Tr. = Transcript of the hearing; BCR = Board-certified radiologist; and B = B reader of x-rays.

[2] At the hearing, the Director's exhibits and Employer's exhibits 1–9 were admitted into evidence without objection. Tr. 4–5. Claimant did not have additional exhibits to admit into the record at the hearing. Tr. 5.

[3] Given the filing date of this claim, subsequent to the effective date of the permanent criteria of Part 718 (*i.e.*, March 31, 1980), the regulations set forth at 20 C.F.R. Part 718 will govern its adjudication. Because the miner's last exposure to coal-mine dust occurred in West Virginia, this claim arises under the jurisdiction of the U.S. Court of Appeals for the Fourth Circuit. *See Broyles v. Director, OWCP*, 143 F.3d 1348, 21 BLR 2-369 (10th Cir. 1998).

[4] The miner's claims 1 and 2 are administratively closed and not subject to adjudication. DX 28, 29.

Board on December 7, 2000. DX 32 at part 25. The miner died during litigation of the claim, on November 9, 2000. DX 1. On December 20, 2000, the miner's widow petitioned for modification of the claim and submitted additional evidence. The District Director issued a Proposed Decision and Order Granting Modification and Awarding Benefits, dated September 20, 2002. DX 1. Employer disagreed with the District Director's determination and requested a formal hearing before the Office of Administrative Law Judges (OALJ) on September 26, 2002. DX 1. The claim was referred to the OALJ on December 24, 2002. DX 40.

The survivor's claim was filed on January 29, 2001. Tr. 8. The District Director issued a Proposed Decision and Order Awarding Benefits dated August 2, 2002. DX 34. Employer disagreed with the District Director's determination and requested a formal hearing before the OALJ. DX 35. The claim was referred to the OALJ on December 24, 2002. DX 40.

The findings of fact and conclusions of law that follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented. Where pertinent, I have made credibility determinations concerning the evidence.

## Background

At the hearing, the miner's widow, Mrs. Jones, testified as follows: She was married to the miner when he died and they had been married for approximately fourteen and a half years. Tr. 9. She has not remarried. *Id.* Mrs. Jones stated that after the miner testified at his hearing in September of 1999, he did not go back to work for any other coal company. Tr. 10. The widow further testified that at the time of the miner's hearing, he did not smoke cigarettes and he did not start smoking again after the hearing. *Id.*

## Medical Evidence

Except as modified or superseded herein, the medical evidence which was set forth in Judge Leland's Decision and Order—Denying Benefits is incorporated by reference herein. The new medical evidence and the medical records submitted in connection with the miner's subsequent modification petition and the survivor's claim are summarized below.

### Chest x-rays

The record contains the following chest x-ray evidence:

| **Exh.#** | **X-ray Date** | **Physician/Qualifications** | **Interpretation** |
|---|---|---|---|
| DX 17 | 7/1/99 | Koppikar | Peripheral interstitial fibrosis. |
| DX 17 | 2/1/00 | Subramanian | Chronic lung changes with acute infiltrates. |

| Exh # | X-ray Date | Physician/Qualifications | Interpretation |
|---|---|---|---|
| DX 1 | 10/9/00 | Wheeler/BCR, B | Negative; no silicosis or CWP, but lung biopsy is needed to diagnose cause of interstitial infiltrate or fibrosis. |
| DX 1 | 10/9/00 | Scott/BCR, B | Negative. Peripheral interstitial fibrosis compatible with UIP. |
| DX 1 | 10/9/00 | Kim/BCR, B | Negative. Interstitial fibrosis in the periphery of lungs, probably UIP or other such as collagen disease. |
| DX 1 | 10/9/00 | Wiot/BCR, B | Negative. IPF, not CWP. |
| DX 1 | 10/9/00 | Spitz/BCR, B | Negative. Bilateral irregular infiltrate at bases and along lateral walls of lung consistent with interstitial fibrosis. |
| DX 1 | 10/9/00 | Shipley/BCR, B | Negative. Advanced interstitial fibrosis. |

CT Scans

The record contains the following CT scan evidence:

| Exh # | Date | Physician | Interpretation |
|---|---|---|---|
| DX 1 | 10/13/00 | Wheeler | No silicosis or CWP. Focal arteriosclerosis left coronary artery. |
| DX 1 | 10/13/00 | Scott | Peripheral linear fibrosis both lungs. Severe, with areas of "honeycomb lung." No evidence of silicosis/CWP. |
| DX 1 | 10/13/00 | Kim | Interstitial fibrosis compatible with either UIP or collagen disease; several small granulomatous calcifications. |
| DX 1 | 10/13/00 | Wiot | No evidence of pneumoconiosis. Emphysematous changes. Basilar interstitial fibrosis with ground-glass character consistent with UIP and IPF. Findings not of coal dust exposure. Insignificant mediastinal adenopathy. |
| DX 1 | 10/13/00 | Spitz | No evidence of CWP. Diffuse pulmonary disease consistent with interstitial fibrosis, etiology unknown. This probably represents IPF. |
| DX 1 | 10/13/00 | Shipley | No findings consistent with CWP. Advanced pulmonary interstitial fibrosis of unknown etiology is present. |

Treatment Records, Medical Reports and Other Evidence

*Logan General Hospital*

The miner was admitted to Logan General Hospital on February 9, 2000 and was discharged on February 19, 2000. The final diagnosis was benign prostatic hypertrophy and the secondary diagnoses were 1) benign prostatic hypertrophy; 2) chronic obstructive pulmonary

disease; 3) diabetes mellitus; 4) atherosclerotic heart disease; 5) status post coronary artery bypass graft (CABG). DX 17.

The miner was admitted to Logan General Hospital again on October 10, 2000 for acute shortness of breath with cyanosis. The diagnoses were 1) severe chronic obstructive pulmonary disease with acute exacerbation with hypoxemia; 2) congestive heart failure; 3) atherosclerotic heart disease, status post CABG in 1998; 4) type II diabetes mellitus; 5) hyperlipidemia; 6) degenerative joint disease. DX 17.

The miner also underwent a lung biopsy at Logan on October 18, 2000. The post-operative impression was "No endobronchial lesions. The biopsies were taken from the posterior and lateral segment of the left lower lobe where the CT changes are significant." DX 17.

The record contains a history and physical exam performed by Dr. Loynab and dated October 27, 2000. The impression was 1) pneumonia, left lower lobe; 2) bronchitis with acute exacerbation; 3) severe chronic obstructive pulmonary disease (COPD); 4) hypertension; 5) hyperlipidemia; 6) diabetes mellitus. DX 17.

The record contains a consultation report of Dr. Kowatli, dated October 27, 2000. Dr. Kowatli's impression was "Respiratory failure. The patient has very advanced pneumoconiosis. A small amount of fluid, bronchitis or asthma can cause him the respiratory failure. The patient's last EKG showed left ventricular hypertrophy most likely secondary to the previous myocardial infarction."

*Dr. Moushmoush*

The record contains a cardiovascular consultation by Dr. Bassam Moushmoush, dated August 30, 2000, the results of a myoview imaging stress test and stress test follow-up report dated September 7, 2000. DX 32–36. Dr. Moushmoush noted that the miner complained of chest pain and shortness of breath. Dr. Moushmoush's assessment was: 1) increasing shortness of breath and chest pain, rule out progressive of coronary artery disease; 2) status post CABG; 3) history of diabetes; 4) history of hyperlipidemia.

The miner's stress test diagnosis was for evaluation of chest pain, post status CABG. Dr. Moushmoush summarized his findings: 1) a fixed lesion exists involving the inferior wall, which could represent old myocardial infarction or diaphragm attenuation; 2) mild ischemia involving the lateral wall; 3) normal contractility of the left ventricle with an ejection fraction of 54%; 4) no EKG changes suggestive of myocardial ischemia; 5) no chest pain during the stress test.

Dr. Moushmoush's assessment of the miner' status following his stress test was: 1) history of chest pain, appears to be stable clinically; 2) history of CABG; 3) history of diabetes; 4) history of hyperlipidemia. He noted that he advised the miner to watch his blood pressure and, because the miner was asymptomatic at the time, the physician would treat him medically.

*Dr. Naeye*

Dr. Richard L. Naeye, board certified in anatomical and clinical pathology, reviewed the miner's medical records and submitted a report dated April 4, 2001. DX 32–33. Dr. Naeye noted that the miner worked in the coal mines for forty and one-half years, with most of his work above-ground at the tipple. Dr. Naeye noted that the miner last worked as a truck driver and that his coal mine employment ended because the mine shut down. Dr. Naeye stated that the miner reported smoking one to one and a half packs of cigarettes per day for thirty-five years. Dr. Naeye noted that the miner complained of exertional dyspnea in 1973 but that pulmonary function studies at that time produced normal values. He further noted that spirometric tests in 1984 revealed no evidence of airway obstruction or parenchymal restriction, but the miner subsequently developed arteriosclerotic coronary artery disease that required quadruple bypass grafts in 1998. Dr. Naeye stated that a 1999 pulmonary function study revealed a mild restrictive abnormality with no airway obstruction; chest x-rays from 1999 did not reveal abnormalities suggesting the presence of CWP. The physician observed that a 1999 x-ray raised the possibility of one or more old calcified granulomas.

Dr. Naeye stated that there are no anthracotic macules, micronodules, or larger deposits of black pigment visible in the lung tissue samples that might be interpreted as evidence of CWP and that the gross lesions described by the autopsy prosector are mainly areas of fibrosis with a small amount of admixed black pigment at a few sites. He stated that there is no free silica in these lesions and the location of black pigment in them is not adjacent to small arteries and airways, which is the characteristic locus of such pigment in CWP. Dr. Naeye opined that some non-occupational disorder caused this extensive fibrosis and that, at some spots, this fibrosis had invaded and destroyed normal lung tissue, engulfing some black pigment in the process. Dr. Naeye stated that this non-occupational disorder and terminal acute lobular pneumonia were responsible for the miner's death. Dr. Naeye concluded that there is no evidence in the lung tissues or in the findings of objective tests that any occupational disorder impaired lung function, caused disability or hastened the death of the miner. Dr. Naeye stated that it is noteworthy that the miner had normal results of pulmonary function studies, arterial blood gas analyses and chest x-rays long after he quit coal mining. Dr. Naeye stated that there is strong published evidence that simple coal workers' pneumoconiosis does not progress after a miner leaves the industry.

Dr. Naeye was deposed on June 25, 2001 and his deposition appears in the record at DX 32. Dr. Naeye testified that he is board certified in anatomic and clinical pathology, that his earlier career consisted of a lot of research on black young disease but that his current focus is on disorders in late term pregnancy. Dr. Naeye testified that he published a series of scientific papers pertaining to CWP in anthracite coal miners in middle Pennsylvania. Dr. Naeye stated that this report was published in the Archives of Pathology and Laboratory Medicine in 1979 and that he was also appointed to a national committee to define the criteria for diagnosing coal workers' pneumoconiosis. Dr. Naeye testified that the standards published in the Archives are still in use today and have never been superseded by any new findings.

Dr. Naeye testified that in reviewing the miner's occupational history, he noted that most or all of the time the miner worked above ground. Dr. Naeye stated that he could not rule out the presence of CWP just because a patient worked above ground, but there are certain things that a

miner does not come in contact with above ground. The physician further stated that if a person does come in contact with these things, the levels are very, very low—the person can get plenty of dust, but the composition of the dust is not the same as the composition of dust underground. Dr. Naeye stated that the miner would still be at risk to develop coal-dust-induced lung disease, but he would be less likely to develop the disease than an underground worker with similar exposure. Dr. Naeye explained that the development of the fibrosis would be a lot less above ground because not much free silica seems to get above ground. He further stated that he considers it "very unusual" to see extensive fibrosis in people who worked above ground.

Dr. Naeye testified that the miner had a significant history of cigarette smoking and that this history is significant in terms of the cardiac disease he developed in later years. Dr. Naeye stated that the miner's 1970 and 1984 ventilatory studies were normal, but the 1999 test showed a mild restrictive abnormality, which does not comport with his cardiac symptoms. Dr. Naeye testified that although some of the CT scans were interpreted as showing diffuse interstitial fibrosis, interstitial fibrosis is not where damage takes place in coal miners—interstitial fibrosis is not part of CWP. Dr. Naeye testified that he was not familiar with stage three pneumoconiosis as a classification for bronchoscopy and has no idea what was being described in the medical report. Dr. Naeye stated that when a patient, like the miner, presents with severe left ventricular hypertrophy the usual causes are chronic heart failure and severe hypertension. Dr. Naeye explained that the miner's lung weights at autopsy are double the normal, which could be a result of terminal heart failure with a lot of fluid. However, in this case, there was fibrous tissue.

Dr. Naeye stated that the fact that there were a lot of pink areas on the pleural surfaces indicates that the amount of black pigment deposited was small and that even with many miners with mild coal workers' pneumoconiosis, most of the cut surface of the lung looks pretty black and it would never be described as gray, as it was here. Dr. Naeye testified that there was some interstitial fibrosis, but explained that one has to be careful in determining whether that is representative or not; pathologists often remove tissue from the worst areas they can find, despite the fact that his committee told pathologists to take "representative" tissues from the lung. Dr. Naeye testified that although the autopsy prosector described characteristic changes of simple CWP in his report, it "blew my mind." He explained that there are no such lesions in the miner's lungs. Dr. Naeye stated that he does not use changes in the lymph nodes to diagnose CWP because, by definition, CWP requires lesions that actually damage lung tissue; what the lymph nodes contain is immaterial. Dr. Naeye stated that exposure to coal mine dust never causes interstitial fibrosis as a primary disorder. He stated that the small amounts of black pigment scattered throughout the dense fibrosis meant that when another disease process appeared in the miner's lungs, it just enveloped areas where there were small amounts of black pigment.

Dr. Naeye testified that he found no evidence of chronic bronchitis or chronic bronchiolitis, which almost every underground coal miner develops. Dr. Naeye testified that he found no changes that would support the prosector's finding of simple CWP with nodular lesions, because the small deposits of black pigment were in a sea of fibrosis that had some other non-occupational cause. He stated that he was certain the fibrosis was non-occupational because he found no free silica crystals at all in this fibrotic process. Dr. Naeye explained that "honeycomb lung" means that the miner had an interstitial disorder, which is completely different than anything coal miners develop. Dr. Naeye stated that the prosector was absolutely

correct in his diagnosis of acute bronchiolitis, which unlike chronic bronchiolitis is part of pneumonia. Dr. Naeye stated that there is so much destruction in the lung tissue provided that he cannot offer an opinion on any changes associated with pulmonary hypertension.

Dr. Naeye concluded that the miner did not have any lifetime pulmonary impairment as a result of chronic coal-mine-induced lung disease because his first two pulmonary function studies were normal, while his last study showed abnormalities compatible with the interstitial disease process. Dr. Naeye testified that the miner's coal mine exposure did not cause or hasten his death and that his death was caused by very severe coronary artery disease, a lot of it related to cigarette smoking damage in the microcirculation of his heart. Dr. Naeye stated that the next important disease was the interstitial process, which may have been the result of viral pneumonia—a common cause of this interstitial process. Dr. Naeye testified that he did not know what caused the fibrosis found in the miner's lungs, but it was not silicosis. Dr. Naeye testified that individual susceptibility is often underestimated as an important factor in the components of CWP that people develop.

Dr. Naeye submitted a rebuttal to Dr. Perper's report dated February 14, 2003. EX 1. Dr. Naeye stated that Dr. Perper failed to distinguish between birefringent crystals comprised of non-toxic silicates and toxic free silica. He explained that there are a few relatively large crystals of non-toxic silicates in the miner's lung, but no very tiny crystals of tissue-damaging free silica. Dr. Naeye explained that Dr. Perper may not understand the significance of the miner's career in a coal preparation plant. He stated that preparation plant workers encounter coal dust but very little, if any, free silica in their working environments. Dr. Naeye stated that Dr. Perper's attribution of the interstitial fibrotic disorder in the lungs of the miner to his exposures to coal mine dust is not credible and any impairments in lung function that may have been present had a fibrogenic origin. Dr. Naeye further opined that the miner's bronchitis and emphysema were too mild to have had a measurable role in hastening his death.

The transcript of Dr. Naeye's second deposition, which took place on March 19, 2003, appears in the record as EX 6.

*Dr. Caffrey*

Dr. Caffrey, board certified in anatomical and clinical pathology, reviewed the miner's medical and autopsy records and reports and wrote an opinion dated May 18, 2001. DX 32 at part 36. Dr. Caffrey's diagnoses were diffuse interstitial pulmonary fibrosis, acute broncho-pneumonia, acute bronchiolitis, and mild-to-moderate amount of anthracotic pigment within lung tissue and hilar lymph-node tissue. Dr. Caffrey noted that the miner worked forty and one-half years in coal mining, most of this work underground but some of it at the surface. Dr. Caffrey noted a discrepancy in the smoking history but concluded that the miner smoked a pack per day beginning at age ten or eleven and stopping in 1969, equal to a thirty-five-pack-year history. Dr. Caffrey stated that he found no CWP or any other occupational pneumoconiosis such as silicosis or asbestosis in the miner.

Dr. Caffrey stated that the autopsy slides show diffuse interstitial pulmonary fibrosis and that there are numerous synonyms for this disease, but the predominant picture is pulmonary

fibrosis. Dr. Caffrey explained that the disease is often thought to be idiopathic because the direct cause cannot be determined. Dr. Caffrey cited a passage of the text *Pulmonary Pathology*, which states that cigarette smoking is a potentially significant factor, although it is infrequently mentioned in discussion concerning pulmonary fibrosis, and that other clinical studies have reported a relatively high frequency of cigarette smoking in persons who develop pulmonary fibrosis. Dr. Caffrey stated that he cannot objectively say that the miner's years of cigarette smoking caused the disease, but it is a possibility. He explained that, in his opinion, because the findings of coal workers' pneumoconiosis or asbestosis are not present on the slides, he definitely does not think that the disease came from coal mine employment.

Dr. Caffrey stated that it is his opinion that the exact cause of the pulmonary fibrosis or interstitial pulmonary fibrosis is not definitely determined. He stated that he does not believe that the necessary findings to make a diagnosis of coal workers' pneumoconiosis, silicosis, or asbestosis are present on the autopsy slides and that he does not believe that coal workers' pneumoconiosis caused the disease from which the miner suffered. Dr. Caffrey further stated that terminally, the miner had bronchopneumonia, which occurred in an individual who was definitely debilitated from a pulmonary standpoint. Dr. Caffrey disagrees with the physician who signed the autopsy report and the cause of death on the death certificate because the physician who filled out the death certificate filled it out before the autopsy report was completed Dr. Caffrey stated that he disagrees with the death certificate diagnosis of "end stage pneumoconiosis" because he is unable to make a diagnosis of simple CWP.

Dr. Caffrey wrote a supplemental opinion dated February 20, 2003. EX 2. He stated that he disagrees with Dr. Perper's diagnosis of "primary diffuse interstitial fibrosis of atypical coal workers' pneumoconiosis" as he is not familiar with that diagnosis and found no reference for it in the "Pathology Standards for Coal Workers' Pneumoconiosis". Dr. Caffrey reiterated Dr. Bush's point that in Dr. Perper's cited reports, only three of the 233 autopsy cases were of coal miners. Dr. Caffrey stated that the miner had evidence of exposure to coal dust, but he did not have the lesions of CWP and he certainly did not have severe pneumoconiosis of the silicotic type cited by Dr. Perper from the text *Pathology of Occupational Lung Disease*. Dr. Caffrey further stated that the miner also had significant heart disease and apparently continued to have chest pain. He stated that the miner had hypertension and diabetes mellitus and that none of these conditions was caused or in any way related to his employment in the coal mine industry. Dr. Caffrey concluded that the opinions given in his original report are still valid and the fact that the miner was a coal miner did not cause, contribute to, or hasten his death.

*Dr. Perper*[5]

The medical report of Dr. Joshua A. Perper is dated October 25, 2001 and appears at DX 32 at part 41. Dr. Perper reviewed the medical records and autopsy materials at the request of the Department of Labor. Dr. Perper noted that the miner worked in the mines for forty and one-

[5] Dr. Perper's credentials are not in the record. As he has appeared before the Office of Administrative Law Judges on many occasions, I take notice that Dr. Perper is Chief Medical Examiner for Broward County, Florida, is licensed to practice medicine in Florida, is board-certified in anatomic and clinical pathology, and holds two faculty appointments at the University of Miami School of Medicine. I obtained this information from the Florida Department of Health website on July 19, 2004 at http://ww2.doh.state.fl.us.

half years and left because the mine shut down. Dr. Perper noted two medical reports by Drs. Ranavaya and Castle, who recorded smoking histories of forty-six and thirty-five pack years respectively. Dr. Perper opined that the miner had evidence of pneumoconiosis based on the following:

1) More than forty years' exposure to coal mine dust.
2) Clinical symptoms of symptomatic respiratory complaints and findings.
3) Evidence of restrictive lung disease and hypoxemia.
4) Evidence of severely abnormal ventilatory studies.
5) Presence of coal workers' pneumoconiosis at autopsy, of the interstitial fibrosis type.
6) It is well known that silica exposure leads to interstitial fibrosis in both clinical and experimental conditions. In recent years, a number of studies have shown an association between diffuse interstitial fibrosis and non-asbestos pneumoconiosis.

Dr. Perper noted that although a number of chest x-ray readers did not observe radiological evidence of coal workers pneumoconiosis, most of the readers recognized the presence of pulmonary interstitial fibrosis. He explained that some readers labeled the fibrosis as "idiopathic" and others raised the possibility of granulomatous disease, even though no granulomatous process was discovered at autopsy. Dr. Perper explained that in the presence of a well recognized fibrogenic agent (coal dust containing silica), it is not reasonable to ascribe the interstitial fibrosis to an unknown fibrogenic process. Dr. Perper further stated that the autopsy disclosed the presence of coal workers' pneumoconiosis, of the atypical interstitial fibrosis type, complicated by acute bronchopneumonia.

Dr. Perper stated that the miner's coal workers' pneumoconiosis was a result of his occupational exposure to coal mine dust. He explained that it is well known that coal workers' pneumoconiosis results from occupational exposure to coal mine dust, and the pathological changes of interstitial type of coal workers' pneumoconiosis included the presence of anthraco-fibrosis with presence of birefringent silica crystals. Dr. Perper stated that cigarette smoking is not associated with interstitial pulmonary fibrosis, that the miner stopped smoking three decades prior to his death, and that pulmonary disease secondary to smoking does not progress once a patient stops smoking. Dr. Perper opined that the miner's long-standing occupational exposure to coal mine dust is clearly consistent and sufficient for the development of coal worker's pneumoconiosis.

Dr. Perper stated that the miner's coal workers' pneumoconiosis was a substantial contributory cause of his death or was a hastening factor in his death. Dr. Perper explained that he reached this conclusion for the following reasons:

1) Coal workers' pneumoconiosis is a well recognized cause of significant pulmonary disability that may lead to early death.
2) The autopsy revealed the presence of a severe type of coal workers' pneumoconiosis of the interstitial fibrosis type.
3) According to the medical records, the miner developed severe respiratory insufficiency and hypoxemia prior to his death, secondary to his coal workers' pneumoconiosis of the interstitial type and complicating bronchopneumonia. Those processes contributed to

death both directly through hypoxemia and indirectly through hypoxemia triggering a fatal arrhythmia in a patient with coronary heart disease.

Dr. Perper stated that Dr. Jarboe's opinion that pneumoconiosis cannot develop past the period of occupational exposure is "clearly incorrect." Dr. Perper explained that recent literature substantiates that the pulmonary damage associated with exposure to coal mine dust and coal workers' pneumoconiosis may progress after the cessation of occupational exposure to silica containing mixed coal mine dust. He illustrated that inhaled silica remains trapped in the lungs and cannot be removed.

Dr. Perper concluded, with a reasonable degree of medical certainty, that:

1) The miner, who had a history of occupational coal-dust exposure of more than thirty years, had evidence of CWP of the interstitial pulmonary fibrosis type and complicating acute bronchopneumonia.
2) The miner's CWP was a result of his long-standing occupational exposure to mixed coal dust containing silica.
3) The miner's CWP of the interstitial pulmonary fibrosis type, and complicating acute bronchopneumonia, was a substantial contributory cause of his death and a hastening factor in his death.
4) Pertinent and reliable medical literature clearly demonstrates that simple CWP may be a cause of severe obstructive lung disease, pulmonary disability and early death, beyond any effect of smoking, and that the disease process is progressive and may advance after cessation of exposure.

*Dr. Bush*

The medical report of Dr. Stephen T. Bush is dated February 1, 2002 and appears at DX 28. Dr. Bush, board certified in anatomic and clinical pathology, reviewed the medical records, reports, death certificate, and autopsy slides. Dr. Bush noted that the miner's smoking history was recorded as one to one and a half packs per day for thirty years and that the miner's above-ground coal mine employment totaled forty years.

Dr. Bush stated that he could respond with reasonable medical certainty that "[t]he lungs show no evidence of coal workers' pneumoconiosis and coal worker macules, micronodules or macronodules are not present." Dr. Bush explained that, if present, these lesions would consist of black dust pigment in macrophages and free in the tissue, forming stellate lesions with fibrosis and surrounding focal dust emphysema. Instead, he noted, the histologic slides show a small amount of black dust pigment (consistent with carbon) associated with relatively few brightly birefringent particles (consistent with silicate mineral). Dr. Bush stated that the dust pigment is diffusely scattered in a few localized areas, particularly in the upper lobes, and the dust pigment is incidentally engulfed in a diffusely fibrotic process extensively involving the lung parenchyma.

Dr. Bush stated that the interstitial fibrosis is more extensive in the right lung than in the left lung. He stated that examination of the left lung shows some areas older than others and

some areas having more lymphocytic cellular accumulation than other areas. He noted that the fibrous tissue in some regions is old and dense and in other regions is loose, fibrillar and less collagenous, indicating a more recent change. Dr. Bush stated that particularly in the lower lobes, the medium sized airways are filled with inflammatory debris and scattered areas of the lung show air spaces filled with recent hemorrhage. Dr. Bush noted a rare organizing thrombus in small blood vessels, particularly in sections from the lower lobes. He also noted that the lymph nodes contain some carbon pigment but are devoid of any fibrotic reaction to the carbon or silicate particles.

As to the remaining questions at issue, Dr. Bush stated that CWP was not present in the miner, that the coal dust present did not contribute in any way to the miner's death, that the miner suffered from a respiratory impairment as a result of diffuse interstitial fibrosis resulting from usual interstitial pneumonia, that the miner was totally disabled by the usual interstitial pneumonia causing extensive interstitial fibrosis of the lungs, that CWP or occupational exposure to coal dust did not contribute to the miner's respiratory impairment or disability, and that neither CWP nor coal mine dust exposure played any role in (or hastened) the death of the miner.

Dr. Bush explained that the coal dust pigment present in the lungs is small in amount and even if one were to consider this pigment evidence of a pneumoconiosis, it would be an extremely mild degree of pneumoconiosis and too limited to have contributed to any impairment.

Further, the physician stated that the dust pigment present was insignificant in degree and in its effect and could not have hastened or played any role in death from the diffuse interstitial lung fibrosis. Dr. Bush disagreed with the death certificate diagnosis of end-stage pneumoconiosis and stated that the pathologic evidence is strongly against such a diagnosis because CWP lesions are not present on the histologic slides and the gross description of the lungs does not indicate CWP. Dr. Bush also disagreed with the autopsy diagnosis of simple coal workers' pneumoconiosis and explained that the twelve histologic slides of lung tissue are an adequate sample to evaluate for coal worker lesions and none are present.

Dr. Bush disagreed with Dr. Perper and stated that Dr. Perper "invents the diagnosis of 'coal workers' pneumoconiosis of the interstitial type' and also causally relates the dust exposure of the miner to the interstitial fibrosis without convincing rationale for doing so." Dr. Bush stated that although Dr. Perper notes the presence of silica crystals, the crystals are actually silicates and are associated only with carbon pigment and not in the other fibrotic areas. He explained that if crystalline silica were distributed in the tissue to a degree that would stimulate fibrosis, one would reasonably expect the lymph nodes to exhibit fibrosis.

Dr. Bush disagrees with Dr. Perper's statement that "it is not reasonable to ascribe the interstitial fibrosis to an unknown pathogenic process" and stated that pulmonary pathology texts indicate that such a statement is unacceptable. He explained that clinically diagnosed idiopathic pulmonary fibrosis occurs in up to twenty-nine out of one hundred thousand male patients, and the most common cause is Usual Interstitial Pneumonia. Dr. Bush further explained that about 75% of IPF patients are current or previous smokers, and the availability of high resolution CT scanning of the lung has greatly improved the ability to distinguish among the causes of IPF—

including the observation that Usual Interstitial Pneumonia causes peripheral basalar interstitial changes. Dr. Bush pointed out that these are the types of changes observed by the reviewers of the miner's CT scans.

Dr. Bush discredited three of Dr. Perper's literature references, explaining that in one study only three of the 233 cases studied were of coal miners, that the authors of the second study point out that pulmonary fibrosis causes confusion regarding causation, and that the author of the third study stated that the findings cannot be generalized and the data available on the study subjects for dust exposure was not very good. Dr. Bush stated that he believes Dr. Perper went beyond reasonable medical certainty in suggesting that dust exposure in a surface coal worker was a cause of the miner's diffuse interstitial pulmonary fibrosis.

*Dr. Spagnolo*

Dr. Spagnolo, who is board certified in internal medicine and pulmonary diseases, reviewed Claimant's medical records and submitted a supplemental report dated March 11, 2003. Dr. Spagnolo's supplemental report appears at EX 3. Dr. Spagnolo's initial report was dated July 31, 1999 and is set forth in Judge Leland's Decision and Order. In his supplemental report, Dr. Spagnolo reiterated that the miner worked in the mines for approximately forty years, that his last job was as a truck driver, and that he quit working when the mine closed. He further noted that the work involved heavy labor but that other physician's reports conflicted as to whether the miner worked at the surface of the mine or underground. Dr. Spagnolo noted that the miner began smoking at age ten or eleven, and smoked a pack of cigarettes daily until 1969. Dr. Spagnolo stated that the medical records revealed that the miner had a history of heart disease, which required a quadruple bypass in 1998, non-insulin dependent diabetes, and several work-related back injuries.

Dr. Spagnolo discussed the medical reports that he reviewed, concluding that none of the supplemental evidence provided any objective basis for him to change his earlier opinion regarding the presence of pneumoconiosis. Dr. Spagnolo stated that in his opinion the miner did not have CWP, based on the entirety of the medical record. He explained that with regard to the chest x-rays, the preponderance of the readings were negative. Dr. Spagnolo stated that the supplemental material indicates that the miner manifested signs and symptoms of a rapidly progressive idiopathic pulmonary fibrosis (IPF). He explained that usual interstitial pneumonia (UIP) is the most common cause of idiopathic pulmonary fibrosis. He further explained that the cardinal features of IPF/UIP include dry cough, exertional dyspnea, end-inspiratory rales, diffuse parenchymal infiltrates on chest x-ray, honeycombing on high-resolution chest tomography, restrictive defect on pulmonary function tests, impaired oxygenation, and exertional dyspnea progressing over months to years. He explained that clubbing is noted in 20–50% of patients and cyanosis and cor pulmonale are late features. Dr. Spagnolo noted that IPF/UIP is more common in males and in current or former smokers. He further stated that the miner had nearly all of these findings.

Dr. Spagnolo stated that he would conclude that as late as March 1999, the miner had essentially normal lung function for his age. He explained that the decrease in the exercise value observed in 1998 may have been related to the miner's compromised cardiac condition, because

the DLCO was still normal in 1999, it is unlikely that the reduced exercise PO2 in 1998 was secondary to an interstitial lung disease problem. Dr. Spagnolo concluded that from a respiratory standpoint, he would expect the miner to have been able to do his regular coal mining or similar work. He further concluded that with the development of IPF/UIP, the miner suffered from reduced respiratory capacity to perform coal mine employment and the miner's progressive IPF/UIP and cardiac disease resulted in his increasing inability to breathe prior to his death. Dr. Spagnolo stated that none of the miner's symptoms, complaints, or medical conditions was related to his coal dust exposure or coal mine employment and, taking into consideration all of the evidence available in this case, his opinion, with a reasonable degree of medical certainty, is that the miner's death was unrelated to and not hastened, even briefly, by pneumoconiosis nor was pneumoconiosis or coal dust exposure a contributing factor in his death.

*Dr. Castle*

Dr. James R. Castle wrote a supplemental report dated March 13, 2003, which appears in the record at EX 4. Dr. Castle initially examined the miner on March 12, 1999 and his report is summarized in Judge Leland's Decision and Order. Dr. Castle is board certified in internal medicine and pulmonary diseases and is a B reader. Dr. Castle stated that the additional medical data he reviewed only served to reinforce his previously stated opinions. He noted that the miner did develop evidence of an interstitial fibrotic process sometime after 1990, as confirmed by radiographic and CT changes. Dr. Castle stated that it was his opinion that the findings were those of idiopathic pulmonary fibrosis, also called usual interstitial pneumonitis. Dr. Castle stated that the miner did not demonstrate any evidence of an obstructive pulmonary process and that, at the time of his examination, he had evidence of a very mild, clinically insignificant degree of restrictive lung disease due to the idiopathic interstitial pulmonary fibrosis or usual interstitial pneumonitis, with no respiratory disability due to that process. Dr. Castle noted that the miner had cardiac problems including congestive heart failure and that he developed episodes of pneumonia requiring hospitalization, with acute pneumonia resulting in his death.

Dr. Castle stated that although the autopsy prosector indicated there was evidence of simple coal workers' pneumoconiosis with nodular lesions, the finding of nodular lesions was not confirmed by the reviewing pathologists, including Dr. Perper. Dr. Castle stated that it continues to be his opinion that the miner did not suffer from CWP and it is his opinion within a reasonable degree of medical certainty that his death was neither caused by, contributed to, nor hastened in any way by coal dust exposure or coal workers' pneumoconiosis. Dr. Castle opined that the miner's death was due to a combination of idiopathic interstitial pulmonary fibrosis with acute bronchopneumonia and acute bronchiolitis. Dr. Castle opined that the miner would have died as and when he did regardless of his occupation as a coal miner.

Dr. Castle was deposed on August 12, 2003. His deposition transcript appears in the record at EX 8.

*Dr. Jarboe*

Dr. Thomas M. Jarboe, board certified in internal medicine and pulmonary disease, submitted a supplemental report dated March 14, 2003 that appears at EX 5. Dr. Jarboe's initial

report was dated August 2, 1999 and is set forth in Judge Leland's Decision and Order. After outlining the medical records and reports he reviewed, Dr. Jarboe stated that within reasonable medical certainty, he does not feel there is sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. He noted that he expressed a similar opinion in his previous report and that this opinion is now bolstered by the availability of the autopsy materials and it is his reasoned opinion that the pathological material does not support a diagnosis of CWP.

Dr. Jarboe opined that the miner did have a significant respiratory impairment, especially prior to his death. He explained that the miner developed idiopathic pulmonary fibrosis, also known as usual interstitial pneumonia, which resulted in significant derangement of gas exchange with severe hypoxemia. Dr. Jarboe noted that the last pulmonary function studies also showed a mild restrictive defect and he feels this mild restriction was caused by the interstitial fibrosis. Dr. Jarboe explained that interstitial pulmonary fibrosis is not caused by coal dust inhalation and that there was no evidence in the miner's tissues that he had CWP. He stated that it is his reasoned opinion that the respiratory impairment the miner developed was due to the interstitial fibrosis and not CWP. Dr. Jarboe also noted that the miner had significant coronary artery disease, which may also have contributed to his respiratory symptoms.

Dr. Jarboe stated that, from a respiratory standpoint, the miner suffered from a reduced capacity to perform his coal mine employment prior to his death. However, Dr. Jarboe stated that he feels this reduced capacity was caused by the interstitial fibrosis and not by coal workers' pneumoconiosis. Dr. Jarboe further opined that, as a whole man, the miner suffered from a reduced capacity to perform his coal mine employment prior to his death but that this reduced capacity was due primarily to the development of interstitial pulmonary fibrosis and coronary artery disease, which are diseases of the general population and were not related to his occupation as a coal miner. Dr. Jarboe opined that the miner's coal dust exposure played no role in any disability he may have had prior to his death. Dr. Jarboe explained that he based this opinion on the fact that autopsy materials clearly showed that any disabling respiratory impairment present was caused by idiopathic pulmonary fibrosis and not by the presence of pneumoconiosis and that the reviewing pathologist emphasized the fact that the amount of dust deposition in the lungs was minimal.

Dr. Jarboe stated that he does not believe that CWP or the miner's coal dust exposure played any role in or hastened his death. He explained that he feels the miner's death was caused by a combination of underlying interstitial fibrosis and superimposed bronchiolitis and bronchopneumonia, and that these conditions were not in any way related to coal dust exposure or the presence of pneumoconiosis. Dr. Jarboe stated that the miner would have died of the same causes and at the same time whether or not he had ever worked as a coal miner.

Dr. Jarboe stated that his opinion regarding the cause of death or the role coal dust exposure played in the miner's disability would not change even if the miner were found to have coal workers' pneumoconiosis. He explained that it is extremely difficult to see how he could be found to have this disorder because there is strong pathological evidence that he did not. Dr. Jarboe stated that there was no evidence the miner had legal pneumoconiosis as the miner's spirometrics prior to the development of fibrosis showed no restriction or obstruction as would be seen in coal dust inhalation. Dr. Jarboe stated that even if someone were to decide that the

miner had coal workers' pneumoconiosis, it is his reasoned opinion that the miner died from underlying interstitial pulmonary fibrosis with superimposed bronchopneumonia and bronchiolitis and that these conditions were not caused by the inhalation of coal dust or the presence of pneumoconiosis.

*Death Certificate*

The miner's death certificate was filed on November 16, 2000 and was completed by N.B. Tuamquin, M.D. DX 32 at part 29. The death certificate lists the immediate cause of death as end-stage pneumoconiosis. Other significant conditions listed are bilateral pneumonia, arteriosclerotic heart disease, type II diabetes mellitus, hypertension, and hyperlipidemia.

*Autopsy Prosector's Report*

An autopsy was performed on December 4, 2000. DX 32 at part 29. The autopsy prosector was Dr. Alex P. Racadag, M.D. The final pathological diagnoses were listed as simple CWP with nodular lesions, end-stage honeycomb lung with diffuse interstitial pneumonia and fibrosis, acute bronchopneumonia; acute bronchiolitis, changes associated with pulmonary hypertension, pulmonary congestion and focal intra-alveolar hemorrhage, and focal small pulmonary organized microthromboemboli. A comment following the diagnoses stated, "The above conditions contributed to the patient's demise."

The clinical abstract stated that the miner was a seventy-seven-year-old white male, retired coal miner, recently discharged after hospitalization for shortness of breath. His chest x-ray showed interstitial lung disease since 1997. He quit smoking in 1969. The patient underwent high resolution CT scan which showed advanced pulmonary fibrosis and underwent bronchoscopy which was positive for black lung. The conclusion at that time was stage III pneumoconiosis with a component of asthma. The patient was seen two days prior to this admission in Dr. Kowatli's clinic. Afterward, the patient developed chills, spitting green sputum, weakness, and shortness of breath, and was seen in the emergency room and found to be severely hypoxic secondary to respiratory failure. He also had severe left ventricular hypertrophy and possible myocardial infarction. He was transferred to the medical floor and, despite treatment, the miner expired. The autopsy was limited to the lungs for black lung purposes by the family's request.

The autopsy of the respiratory system revealed that the left lung weighed 680 grams while the right lung weighed 790 grams. The pleural surfaces were predominantly gray-black with interspersed tan-pink areas. The right lower lobe was bulkier than the rest of the other lobes. Serial sectioning showed a tan-gray cut surface with scattered black-gray mottling. Scattered firm areas were noted, particularly in both lower lobes, with tan-gray cut surface. Small palpable black nodules were noted, measuring up to 0.4 cm in maximum dimension. Emphysematous changes were noted, particularly in the subpleural areas, representing empty spaces measuring up to 0.5 cm in maximum dimension, which also formed a honeycomb pattern within the parenchyma. No large bullae or masses were identified. The hilar lymph nodes had a black to gray solid cut surface. The bronchial tree contained a minimal amount of gray mucus. The blood vessels were filled with post-mortem blood clots.

The microscopic examination of sections from all the lobes of both lungs showed similar changes. There was severe interstitial fibrosis and widening with lymphocytic and neutrophilic infiltrates. There were dilated alveolar and bronchiolar spaces plugged with inspissated mucus and numerous neutrophils. There was also focal hemorrhage. There was thickening of the medium-sized blood vessel walls associated with smooth muscle hyperplasia and few medium-sized vessels were noted to have organized thrombi. Many bronchioles were cystically dilated with a honeycomb pattern, and interalveolar septa were lined by hyperplastic often atypical pneumocytes II. Scattered coal macules and nodules were noted composed of aggregates of black pigmented macrophages with moderate collangenized fibrosis and focal emphysema around bronchioles and small blood vessels. Few intra-alveolar brown pigmented macrophages were also present. There were also focal intra-alveolar hemorrhages. These changes were more pronounced on the right lung. Focal osseous metaplasia was noted. The hilar lymph nodes likewise showed black pigmented microphages with some fibrosis.

*Dr. Tomashefski*

Dr. Joseph F. Tomashefski, board certified in anatomical and clinical pathology, reviewed the miner's records and medical reports and wrote an consultation opinion dated April 17, 2002. DX 32. Dr. Tomashefski noted that the miner worked mainly above ground for approximately forty years and that he smoked cigarettes a cumulative total of about thirty-five pack years. Dr. Tomashefski stated that his review of the autopsy slides revealed diffuse interstitial fibrosis with dilated, respiratory epithelial-lined airspaces, imparting a honeycomb pattern—a pattern most developed in the lower lobes. He noted that in the upper lobes, and focally in the lower lobes, there is an alveolar septal pattern of active fibroplasia, mild chronic interstitial inflammation, organizing hyaline membranes and diffuse type II pneumocyte hyperplasia. Dr. Tomashefski stated that other incidental histologic features include focal acute alveolar macrophages, rare recent pulmonary emboli, and acute mucopurulent exudate in dilated airspaces. He stated that in the right lung hilum is a small, fibronecrotic granuloma and that the hilar lymph nodes are congested and contain fine black pigment.

Dr. Tomashefski noted that throughout the lung sections there is a small amount of fine, black pigment deposited in the pleura and around blood vessels and this amount of pigment is comparable to that which might be seen in a non-mining urban dweller. He stated that neither coal macules, micronodules, nor significant birefringent mineral particles are seen.

Dr. Tomashefski stated that, in his opinion, the miner had end-stage fibrotic lung due to idiopathic pulmonary fibrosis. He stated that although the autopsy was limited to the lungs, it is reasonable to assume that idiopathic pulmonary fibrosis was the underlying cause of death. He stated that the immediate cause of death was likely respiratory failure precipitated by acute prurulent bronchitis and pneumonia, pulmonary emboli, and organizing diffuse alveolar damage, which may represent the accelerated phase of idiopathic pulmonary fibrosis. Dr. Tomashefski explained that because he did not see any coal macules, it is also his opinion that the miner did not have simple CWP and that the pattern of interstitial fibrosis is not that of complicated pneumoconiosis. Dr. Tomashefski stated that there is no indication whatsoever that the miner's diffuse interstitial fibrosis was due to coal dust exposure and that there is minimal pigment observed in his lung tissue. He explained that, by definition, the cause of idiopathic pulmonary

fibrosis is not known, but it is not considered to be caused by coal dust exposure, especially in the absence of even simple CWP or significant particulates in the fibrotic tissue.

Dr. Tomashefski disagreed with Dr. Perper's observations and conclusions. He stated that the miner does not meet the histologic criteria for coal workers' pneumoconiosis as proposed in the pulmonary standards for CWP or in a more recent pathology textbook. He stated that the role of coal dust in causing diffuse interstitial fibrosis is controversial and in the miner's case, the lack of association of interstitial fibrosis with significant dust deposition within the lung excludes mineral dust as a cause of his severe, fatal diffuse interstitial fibrosis. Dr. Tomashefski stated that Dr. Perper's statement that simple CWP may be a cause of severe obstructive lung disease is irrelevant because the miner did not have evidence of obstructive lung disease by pulmonary function tests, and had no anatomic evidence of CWP in his lungs at autopsy.

Dr. Tomashefski concluded that the miner's disabling lung disease was not caused or influenced by his coal mining occupation and he would have died at approximately the same time and in the same manner, even if he had never been a coal miner.

*Dr. Crouch*

Dr. Erika C. Crouch reviewed the miner's medical records and reports and wrote a report dated May 22, 2002. Dr. Crouch is board certified in anatomic pathology. DX 32 at part 46. Dr. Crouch noted that the slides contain sections of pulmonary tissue and bronchopulmonary or hilar lymph nodes. She stated that the lungs are remarkable for diffuse chronic organizing interstitial pneumonia with areas of dense fibrosis and honeycombing. Dr. Crouch noted that many of the areas appear relatively homogeneous in terms of the age of fibroproliferative lesions. She stated that multiple areas of acute bronchopneumonia are noted and one section shows recent organizing pulmonary thromboemboli. Dr. Crouch stated that there is relatively mild deposition of irregular black to brown particles, some of which is consistent with coal dust. She explained that although coal dust is sometimes entrapped in areas of fibrosis, no coal dust macules, micronodules, nodules, or areas of massive fibrosis are observed. Dr. Crouch stated that a few hyalinized nodular lesions consistent with inhalation of coal dust containing silica are identified in the lymph nodes; however, no typical silicotic nodules are identified within the lung parenchyma. She noted that polarization microscopy reveals only relatively small numbers of short needle-like birefringent particles consistent with silicates within the lung. She stated that there is no concordance between the sites or severity of dust deposition and the extent or severity of the observed fibroproliferative changes.

Dr. Crouch's diagnoses were chronic organizing interstitial pneumonia, acute bronchopneumonia, recent thromboemboli, and mild coal dust deposition. Dr. Crouch stated that there is histologic evidence of coal dust deposition within the lungs, but there is no histologically discernible coal workers' pneumoconiosis. She stated that although there is non-specific trapping of dust within some areas of fibrosis, there is no association between the deposited dust and the presence of fibrosis. Dr. Crouch stated that diagnostic changes of pneumoconiosis or simple silicosis are not evident. She stated that trapping of inhaled dust in areas of fibrosis is a common and entirely anticipated finding that is often observed in the lungs of individuals with lung fibrosis and no occupational history of dust exposure.

Dr. Crouch stated that the etiology of the observed fibrosis is uncertain, but the histologic features do not support a role of inhaled dust. She explained that some features including the observed temporal heterogeneity are consistent with usual interstitial pneumonia as observed in idiopathic pulmonary fibrosis. Dr. Crouch stated that her impression is that the lungs show a relatively old fibrosing process with associated honeycombing, with a superimposed more recent and temporally homogenous fibroproliferative reaction. Dr. Crouch explained that clinical evidence that the disease is more severe on the right is also highly atypical for the diagnosis of idiopathic pulmonary fibrosis and, given the miner's advanced age, the possibility of aspiration pneumonia, which more commonly involves the right lung, should be considered even though no definitive histologic evidence of aspiration was observed.

Dr. Crouch stated that occupational coal dust exposure could not have caused any clinically significant degree of respiratory impairment or disability and could not have caused, contributed to, or otherwise hastened the miner's death. She stated that the immediate cause of death cannot be established with certainty due to the limitations of autopsy to the lungs. She stated that death may have resulted from bronchopneumonia, which is unrelated to the dust deposition.

## Miner's Claim

### Length of Coal Mine Employment

The parties have stipulated and I find that the miner was a coal miner within the meaning of the Act for at least 23 years. Tr. 6.

### Date of Filing

The parties have stipulated and I find that Claimant filed his claim for benefits under the Act on April 9, 1998. Tr. 7.

### Responsible Operator

The parties have stipulated and I find that Elkay Mining Company is the responsible operator and will provide payment of any benefits awarded to Claimant. Tr. 7.

### Dependents

The parties have stipulated and I find that Claimant has one dependent, his wife Frances, for purposes of augmentation of benefits under the Act. Tr. 7.

Modification and Duplicate Claim

The parties stipulated at the hearing that the current claim was filed more than one year after the denial of Claimant's previous claim for benefits. As such, he must establish that there has been a material change in conditions since the denial of the last claim. 20 C.F.R. § 725.309 (d) (2000). The administrative law judge must consider all of the new evidence, favorable and unfavorable, and determine whether the miner has proven at least one of the elements of entitlement previously adjudicated against him. If the miner establishes the existence of that element, he has demonstrated a material change in condition and all of the record evidence must then be evaluated to determine if he is entitled to benefits under the Act. *Labelle Processing v. Swarrow*, 72 F.3d 308 (3rd Cir. 1995); *Lisa Lee Mines v. Director, OWCP*, 86 F.3d 1358 (4th Cir. 1996).

The parties further stipulated that this claim is also a modification of the miner's claim because the widow filed her request for modification within one year after the denial of the claim. The regulations provide that upon the showing of a "change in conditions" or a "mistake in a determination of fact," the terms of an award or the decision to deny benefits may be reconsidered. 20 C.F.R. § 725.310 (2000). As with a duplicate claim, if a change in condition is established, a *de novo* review of the record will be conducted to determine if Claimant is entitled to benefits. *See Kovac v. BCNR Mining Corp.*, 14 B.L.R. 1-156 (1990), *aff'd on recon.*, 16 B.L.R. 1-71 (1992); *Dingess v. Director, OWCP*, 12 B.L.R. 1-141 (1989); *Cooper v. Director, OWCP*, 11 B.L.R. 1-95 (1988). In addition, even if a "change in conditions" is not established, evidence must be considered to determine whether a "mistake in a determination of fact" was made, even where no specific mistake of fact has been alleged. *See O'Keeffe v. Aerojet-General Shipyards, Inc.*, 404 U.S. 254, 257 (1971); *Jessee v. Director, OWCP*, 5 F.3d 723 (4th Cir. 1993). In reviewing the evidence of record, both old and new, I find that Judge Leland did not make a mistake in a determination of fact with the evidence that was before him at that time.

Claimant's previous claim was denied because the evidence did not establish the existence of pneumoconiosis arising out of coal mine employment or total disability due to pneumoconiosis. Thus, in order for Claimant to prove a change in conditions, the new evidence must be evaluated to determine whether these elements can now be established.

Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence and draw his/her own conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 B.L.R. 1-190 (1989); *Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986); *Todd Shipyards Corp. v. Donovan*, 300 F.2d 741 (5th Cir. 1962). The adjudicator's function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty*; *Fagg v. Amax Coal Co.*, 12 B.L.R. 1-77 (1988), *aff'd*, 865 F.2d 916 (7th Cir. 1989); *Short v. Westmoreland Coal Co.*, 10 B.L.R. 1-127 (1987); *Piccin v. Director, OWCP*, 6 B.L.R. 1-616 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266 (7th Cir. 1983).

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. *See Wright v. Director, OWCP*, 7 B.L.R. 1-475 (1984); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984); *Crider v. Dean Jones Coal Co.*, 6 B.L.R. 1-606 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983); *see also Stevenson v. Windsor Power House Coal Co.*, 6 B.L.R. 1-1315 (1984). The weight of the evidence, and determinations concerning credibility of medical experts and witnesses, however, is for the administrative law judge. *Mabe v. Bishop Coal Co.*, 9 B.L.R. 1-67 (1986); *Brown v. Director, OWCP*, 7 B.L.R. 1-730 (1985); *see also Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Henning v. Peabody Coal Co.*, 7 B.L.R. 1-753 (1985); *Peabody Coal Co. v. Benefits Review Board*, 560 F.2d 797, 1 B.L.R. 2-133 (7th Cir. 1977).

As the trier-of fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof. *Kuchwara v. Director, OWCP*, 7 B.L.R. 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. *See White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

Pneumoconiosis

Section 718.202 provides that the existence of pneumoconiosis can be established by chest x-ray, autopsy or biopsy evidence, the presumptions in §§ 718.304, 718.305, and 718.306, and medical opinions finding that Claimant has pneumoconiosis as defined in § 718.201.[6] *See* § 781.202(a)(1)–(4). All types of relevant evidence must be weighed together under § 718.202(a) in assessing whether the miner suffers from pneumoconiosis. *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th. Cir. 2000).

*Chest x-ray Evidence*

The interpretations of physicians who are dually qualified (as board-certified radiologists and B-readers) are entitled to the greatest weight. The Benefits Review Board held that it is proper to credit the interpretation of a dually qualified physician over the interpretation of a B-reader. *Cranor v. Peabody Coal Co.*, 22 B.L.R. 1-1 (1999). Of the x-ray evidence submitted in connection with the most recent claim, there were eight interpretations of three x-rays.

None of the physicians interpreted the x-rays as positive for pneumoconiosis and the interpretations of the most recent x-ray, taken October 9, 2000, were uniformly negative for pneumoconiosis by six dually qualified physicians: Drs. Wheeler, Scott, Kim, Wiot, Spitz, and Shipley. Dr. Koppikar, who is neither a board-certified radiologist nor a B-reader, noted that the July 1, 1999 x-ray revealed "peripheral interstitial fibrosis." Dr. Subramanian, who is also neither a board-certified radiologist nor a B-reader, commented that the February 1, 2000 x-ray revealed "chronic lung changes with acute infiltrates."

[6] 20 C.F.R. § 718.201 defines pneumoconiosis as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment.

I conclude that Claimant has failed to establish the existence of pneumoconiosis by a preponderance of the chest x-ray and CT scan evidence, pursuant to 20 C.F.R. §718.202(a)(1).

*CT Scan Evidence*

The record also contains six interpretations of a CT scan taken on October 13, 2000. Five of the six physicians interpreted the CT scan as negative for pneumoconiosis.

*Lung Biopsy*

The miner underwent a lung biopsy at Logan General Hospital on October 18, 2000. The results showed no endobronchial lesions. "The biopsies were taken from the posterior and lateral segment of the left lower lobe where the CT changes are significant." DX 17.

*Post-Mortem Evidence*

The death certificate was completed by N.B. Tuamquin, M.D and lists the immediate cause of death as end-stage pneumoconiosis. A death certificate, in and of itself, is an unreliable report of the miner's condition. *Smith v. Camco Mining, Inc.*, 13 B.L.R. 1017 (1989); *Addison v. OWCP*, 11 B.L.R. 1-68 (1998). It is unknown on what information Dr. Tuamquin based his information and I note that he included several other substantial diseases, such as bilateral pneumonia and heart disease, as other significant conditions. In addition, the mere fact that a death certificate refers to pneumoconiosis cannot be viewed as a reasoned medical finding, particularly if, as in this case, no autopsy has been performed. *Lango v. Director, OWCP*, 104 F.3d 573 (3d Cir. 1997). *See also Bill Branch Coal Co. v. Sparks*, 213 F.3d 186 (4th Cir. 2000) (a death certificate stating that pneumoconiosis contributed to a miner's death, without some further explanation, is insufficient). Based on the foregoing, the contents of the death certificate cannot be considered a reasoned medical finding and I afford the death certificate little weight.

Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). The autopsy prosector, Dr. Racadag, listed the final pathological diagnoses as: simple coal workers' pneumoconiosis with nodular lesions; end-stage honeycomb lung with diffuse interstitial pneumonia and fibrosis; acute bronchopneumonia; acute bronchiolitis; changes associated with pulmonary hypertension; pulmonary congestion and focal intra-alveolar hemorrhage; focal small pulmonary organized microthromboemboli. A comment following the diagnoses stated, "the above conditions contributed to the patient's demise." Dr. Racadag did not explain the relative contributions of the diagnosed conditions to the miner's death. Without a reasoned analysis to this effect, the report lacks sufficient reasoning to be of assistance to me in my analysis of the evidence. I therefore afford the prosector's report little weight.

*The Presumptions*

Under 20 C.F.R. §718.202, it shall be presumed that a miner is suffering from pneumoconiosis if the presumptions set forth in §§ 718.304, 718.305, or 718.306 apply. Initially, I note that the miner cannot qualify for the § 718.304 presumption because there is no

credible evidence that he suffered from complicated pneumoconiosis. Claimant does not qualify for the § 718.305 and § 718.306 presumptions because he filed his claim after January 1, 1982.

*Medical Opinion Evidence*

Additionally, a determination of the existence of pneumoconiosis may be made if a physician, exercising sound medical judgment, based upon certain clinical data and medical and work histories and supported by a reasoned medical opinion, finds that the miner suffered from pneumoconiosis notwithstanding a negative x-ray. 20 C.F.R. § 718.202(a)(4)(2000). Medical reports based upon and supported by patient histories, a review of symptoms, and a physical examination constitute adequately documented medical opinions as contemplated by the Regulations. *Justice v. Director, OWCP*, 6 B.L.R. 1-1127 (1984). However, where the physician's report, although documented, fails to explain how the documentation supports its conclusions, an administrative law judge may find the report is not a reasoned medical opinion. *Smith v. Eastern Associated Coal Co.*, 6 B.L.R. 1-1130 (1984). A medical opinion shall not be considered sufficiently reasoned if the underlying objective medical data contradicts it. *White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

The reports of nine physicians were submitted regarding the miner's medical condition. Drs. Naeye, Caffrey, Bush, Spagnolo, Castle, Jarboe, Tomashefski, and Crouch concluded that Claimant did not have CWP and Dr. Perper concluded that Claimant did have coal workers' pneumoconiosis. Drs. Naeye, Caffrey, Perper, Bush, Tomashefski and Crouch are pathologists and Drs. Spagnolo, Castle, and Jarboe are pulmonologists.

Dr. Naeye, a board certified pathologist who is on the faculty at the Penn State University College of Medicine, was appointed to the original committee of the American College of Pathologists to identify the components of coal workers' pneumoconiosis. Dr. Naeye opined that some non-occupational disorder and a terminal acute lobular pneumonia were responsible for the miner's disability and death. Like the other physicians, Dr. Naeye diagnosed interstitial fibrosis but opined that it was not occupationally related because he could not find any free silica crystals in the fibrotic process. In addition, Dr. Naeye stated that he did not find any changes that would support the prosector's finding of simple CWP. In fact, he noted a lot of pink areas on the pleural surfaces, indicating that the amount of black pigment deposited was small, while in many miners with mild CWP, most of the cut surface of the lung looks black. Finally, Dr. Naeye stated that it is noteworthy that the miner's objective tests, including chest x-rays, pulmonary function studies, and arterial blood gas studies, were normal long after he quit coal mining and that there is strong published evidence that simple CWP does not progress after the miner leaves the industry.

While I find that Dr. Naeye's opinion is well documented, I note that the Fourth Circuit—in whose jurisdiction this case arises— as well most of the other Circuit Courts and the Benefits Review Board hold that pneumoconiosis is a progressive and irreversible disease. *Lane Hollow Coal v. Lockhart*, 137 F.3d 799, 803 (4th Cir. 1992); *Barnes v. Mathews*, 562 F.2d 278, 279 (4th Cir. 1977) (pneumoconiosis is a slow, progressive disease often difficult to diagnose at early stages); *Peabody Coal Co. v. Odom*, 342 F.3d 486 (6th Cir. 2003) (pneumoconiosis is a progressive and latent disease which can arise and progress even in the absence of continued

exposure to coal dust); *Labelle Processing v. Swarrow*, 72 F.2d 308 (3rd Cir. 1996); *Faulk v. Peabody Coal Co.*, 14 B.L.R. 1-18 (1990); *Andryka v. Rochester & Pittsburgh Coal Co.*, 14 B.L.R. 1-34 (1990). As Dr. Naeye's opinion directly contradicts established case law, I accord his opinion little weight.

Dr. Caffrey, a board certified pathologist, opined that he is unable to make a diagnosis of CWP or any other occupational pneumoconiosis such as silicosis or asbestosis in the miner, and that the predominant picture is that of pulmonary fibrosis. Dr. Caffrey's opinion, however, is inconsistent. Although he notes the presence of mild to moderate anthracotic pigment identified within lung tissue and hilar lymph node tissue, he does not explain why he excludes this anthracotic pigment as a possible cause of the pulmonary fibrosis. Instead, Dr. Caffrey states that it is his opinion that the exact cause of the pulmonary fibrosis or interstitial pulmonary fibrosis is not definitely determined and that he does not believe that the necessary findings to make a diagnosis of coal workers' pneumoconiosis, silicosis, or asbestosis are present on the autopsy slides. Dr. Caffrey never discusses the possibility of anthracosis—despite having noted the anthracitic pigment—yet is thorough enough in his elimination of silicosis and asbestosis. Therefore, I find that Dr. Caffrey's opinion is not well reasoned and is entitled to less weight.

Dr. Perper, a pathologist, concluded that the miner had evidence of coal workers' pneumoconiosis of the interstitial pulmonary fibrosis type, complicating acute broncho-pneumonia, and that CWP was a result of his long-standing occupational exposure to mixed coal dust containing silica. Dr. Perper based his conclusions on the miner's more than forty years of exposure to coal dust, symptomatic respiratory complaints and findings, restrictive lung disease and hypoxemia, and severely abnormal ventilatory studies. While Dr. Perper noted that the miner worked for more than forty years, he does not discuss the fact that the miner's work was primarily above ground, nor does he indicate that he is aware of or considered the miner's level of dust exposure.[7] In addition, Dr. Perper's statement of "severely abnormal" ventilatory studies goes against the weight of the objective evidence. All of the miner's pulmonary function studies were normal, with the exception of the last study which, by all other accounts, revealed a mild restrictive defect but nothing "severely abnormal." In addition, as Dr. Tomashefski noted, Dr. Perper's opinion that simple CWP may be a cause of severe obstructive lung disease is irrelevant, as the miner did not have evidence of obstructive lung disease.

Finally, Dr. Perper's diagnosis of "severe coal workers' pneumoconiosis of the interstitial pulmonary fibrosis type" is confusing without further explanation. Dr. Caffrey stated that he is not familiar with that diagnosis and Dr. Bush went so far as to state that Dr. Perper "invents" the diagnosis. Although I suspect that Dr. Perper is implying that the pulmonary fibrosis is a result of some form of "legal" pneumoconiosis, I am not willing or able to make that conjecture.[8] For the above reasons, I find that Dr. Perper's opinion is not well reasoned and is entitled to little weight.

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[7] I note that the miner testified before Judge Leland that he worked at the surface of the mines, and that he made no testimony whatsoever about the amount of dust he was exposed to during his coal mine employment.

[8] The Fourth Circuit has issued a number of decisions addressing clinical versus legal pneumoconiosis. *See Barber v. Director*, OWCP, 43 F.3d 899 (4th Cir. 1995); *Dehue v. Director*, OWCP, 65 F.3d 1189 (4th Cir. 1995); *Hobbs v. Clinchfield Coal Co.*, 45 F.3d 819 (4th Cir. 1995).

Dr. Bush, a board certified pathologist, opined that the miner's lungs show no evidence of coal workers' pneumoconiosis, and coal worker macules, micronodules, or macronodules are not present. In reaching his conclusion, Dr. Bush noted that although the histologic slides show a small amount of black dust pigment consistent with carbon, and the lymph nodes contain some carbon pigment, the coal dust present was insignificant in degree and the lymph nodes are devoid of any fibrotic reaction to the carbon or silicate particles. Moreover, Dr. Bush stated that if crystalline silica were distributed in the tissue to a degree that would stimulate fibrosis, one would reasonably expect the lymph nodes to exhibit fibrosis. Dr. Bush also explained that high-resolution CT scanning of the lung has greatly improved the ability to distinguish among the causes of interstitial pulmonary fibrosis, and that usual interstitial pneumonia causes peripheral basalar interstitial changes, which were the changes observed by reviewers of the miner's CT scan. Dr. Bush explained that clinically diagnosed idiopathic pulmonary fibrosis occurs in up to twenty-nine out of one hundred thousand male patients, and that the most common cause is usual interstitial pneumonia and about 75% of patients are current or previous smokers. Dr. Bush also considered the miner's work and social history in arriving at his conclusion. As such, I find that Dr. Bush's opinion is well-documented and his opinion, which took into consideration the objective findings including the CT scan evidence, is better supported by the objective medical data of record. Therefore, I accord his opinion greater weight.

Dr. Spagnolo, who is a board certified pulmonologist, opined that the miner did not have coal workers' pneumoconiosis based on the entirety of the medical record. He explained that the preponderance of x-rays were negative and that the records indicate that the miner manifested signs and symptoms of a rapidly progressive idiopathic pulmonary fibrosis. Dr. Spagnolo explained that the most common cause of idiopathic pulmonary fibrosis is usual interstitial pneumonia and that the main features include dry cough, exertional dyspnea, end-inspiratory rales, diffuse parenchymal infiltrates on chest x-ray, honeycombing on high resolution CT scan, restrictive defect on pulmonary function tests, impaired oxygenation, exertional dyspnea progressing over months or years, clubbing of the digits, cyanosis, and cor pulmonale. He explained that the condition is more common in males and in current or former smokers and that the miner manifested almost all of these findings. In arriving at his opinion, Dr. Spagnolo took into consideration the miner's occupational and social histories, and the objective medical evidence, in addition to the autopsy slides and other physician's reports. Therefore, I find that his opinion is well-documented and, because it is supported by the objective medical data of record, it is well-reasoned and is entitled to great weight.

Dr. Castle, a board certified pulmonologist, opined that the miner suffered from idiopathic pulmonary fibrosis or usual interstitial pneumonitis. He explained that although the autopsy prosector indicated that there was evidence of simple CWP with nodular lesions, the finding of nodular lesions was not confirmed by any of the reviewing pathologists, including Dr. Perper. Dr. Castle also reiterated these points in his deposition. Dr. Castle stated that it continues to be his opinion that the miner did not suffer from coal workers' pneumoconiosis. Although Dr. Castle reviewed all of the miner's records and actually examined the miner at one time, administering a pulmonary function study, I do not consider his opinion to be well reasoned. In neither his medical report nor his deposition testimony does he explain how he reached his opinion. Unlike Drs. Bush and Spagnolo, Dr. Castle simply reiterates that the miner

suffered from idiopathic pulmonary fibrosis, without fully explaining on what evidence he based his opinion. Accordingly, I find that his opinion is entitled to less weight.

Dr. Jarboe, who is a board certified pulmonologist, opined that there is not sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. Dr. Jarboe opined that the miner developed idiopathic pulmonary fibrosis, also known as usual interstitial pneumonia. Dr. Jarboe based his opinion on the fact that the autopsy materials showed no presence of pneumoconiosis and that the reviewing pathologist emphasized that the amount of dust deposition in the lungs was minimal. Dr. Jarboe also stated that there was no evidence the miner had legal pneumoconiosis because the spirometrics prior to the development of the fibrosis showed no restriction or obstruction as would be seen in coal dust inhalation. Like Dr. Castle, Dr. Jarboe does not explain the underlying bases for his diagnoses in relation to the objective medical evidence; therefore, his opinion is not well reasoned. I find that Dr. Jarboe's opinion is entitled to little weight.

Dr. Tomashefski, a board certified pathologist, opined that the miner had end-stage fibrotic lung disease due to idiopathic pulmonary fibrosis. He explained that although there is a small amount of fine black pigment deposited in the pleura, this amount of pigment is comparable to that which might be seen in a non-mining urban dweller, and that neither coal macules, micronodules, nor significant birefringent particles are seen. Dr. Tomashefski explained that because he did not see any coal macules, it is his opinion that the miner did not have simple coal workers' pneumoconiosis and the pattern of interstitial fibrosis is not that of complicated pneumoconiosis. He opined that there is no indication whatsoever that the miner's diffuse interstitial fibrosis was due to coal dust exposure, as there is minimal pigment observed in the lung tissue. Dr. Tomashefski concluded that the lack of association of interstitial fibrosis with significant dust deposition within the lung excludes mineral dust as the cause of his severe, fatal, diffuse interstitial fibrosis. While Dr. Tomashefski did not discuss his findings in relation to the majority of the objective medical evidence, he did explain the basis for his opinion based upon the autopsy evidence. Therefore, I find that his opinion is entitled to greater weight than those of Drs. Naeye, Caffrey, Perper, Castle, and Jarboe but less weight than those of Drs. Bush and Spagnolo.

Dr. Crouch, who is a board certified pathologist, diagnosed chronic organizing interstitial pneumonia, acute bronchopneumonia, recent thromboemboli, and mild coal dust deposition. She opined that although there is histologic evidence of coal dust deposition within the lungs, there is no histologically discernible coal workers' pneumoconiosis, and even though there is non-specific trapping of dust within some areas of fibrosis, there is no association between the deposited dust and the presence of fibrosis. Dr. Crouch explained that trapping of inhaled dust in areas of fibrosis is a common and entirely anticipated finding that is often observed in the lungs of individuals with lung fibrosis and no occupational history of dust exposure. I find that Dr. Crouch's opinion is not well-reasoned in that she does not explain why the coal dust deposits could not be a contributor of the fibrosis. I note that she explains that dust in areas of fibrosis is a common finding in people with no occupational exposure, yet she does not discuss the role that occupational exposure may have had in this case. In addition, her report does not indicate whether she considered or was aware of the miner's work and social histories. Thus, I find that her opinion is not well-documented or well-reasoned and is entitled to less weight.

I find that the medical opinions of Drs. Bush, Spagnolo, and Tomashefski to be the best reasoned opinions and that the opinions of Drs. Bush and Spagnolo best synthesize all of the medical evidence of record. In addition, I find that the opinion of Dr. Perper, the only physician who concluded that the miner suffered from pneumoconiosis, is not well reasoned in comparison to the contrary opinions of Drs. Bush, Spagnolo, and Tomashefski. Accordingly, I find that Claimant has not met his burden of establishing the existence of pneumoconiosis via the medical opinion evidence pursuant to 20 C.F.R. § 718.202(a)(4)(2000).

*Weighing All Evidence Together*

Pursuant to the holding in *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), I must weigh all of the evidence under § 718.202(a) together in order to make a determination regarding the existence of pneumoconiosis. I found previously that Claimant was unable to establish its existence through the chest x-ray evidence, that the presumptions at § 718.202 were inapplicable to the facts in this matter, and that the conclusions of the better reasoned medical opinions did not establish the existence of pneumoconiosis pursuant to § 718.202(a)(4). Accordingly, weighing all of the evidence together, I find that Claimant has not established the existence of pneumoconiosis, one of the elements previously adjudicated against him, pursuant to § 718.202(a). As Claimant did not establish that he suffers from pneumoconiosis, he cannot establish that pneumoconiosis arose from coal mine employment.

Evidence of Total Disability

Claimant's previous claim was also denied because he failed to prove that he was totally disabled due to pneumoconiosis. Total disability is defined as pneumoconiosis which prevents or prevented a miner from performing his usual coal mine employment or other comparable gainful work. 20 C.F.R. §§ 718.305(c), 718.204(b)(1)(2). A finding of total disability may be based on the criteria found in § 718.204(c)(1)–(5).

Subsection § 718.204(c)(1) provides that total disability may be established by pulmonary function testing. There are no pulmonary function studies submitted in connection with the current claim; however, during the course of this litigation, the record contains the results of eight pulmonary function studies, none of which produced qualifying results. Accordingly, I find that the miner has failed to establish total disability by pulmonary function evidence pursuant to § 718.204(c)(1).

Subsection 718.204(c)(2) provides that qualifying arterial blood gas testing may establish total disability. There are six arterial blood gas studies in the record, although none were submitted with the current claim. Most of the arterial blood gas studies resulted in qualifying values. Accordingly, I find that the miner has established total disability by the arterial blood gas study evidence.

There is no evidence that the miner suffered from cor pulmonale with right-sided congestive heart failure, thus total disability is not established under § 718.204(c)(3).

The Claimant may also establish total disability if a physician, exercising reasoned medical judgment and based on medically acceptable clinical and laboratory techniques, concludes that the miner's respiratory or pulmonary impairment prevented him from engaging in his usual coal mine work or in comparable and gainful employment. 20 C.F.R. § 718.204(c)(4). Of the nine physicians rendering medical opinions, Drs. Bush, Spagnolo, and Jarboe concluded that the miner was impaired enough to be unable to perform his previous coal mine employment. Dr. Naeye did not conclude that the miner was too impaired from a pulmonary standpoint from returning to his usual work and Dr. Castle opined that although the miner's last pulmonary function study showed a slight restrictive element, it was a result of his impaired cardiac function from his heart disease. Drs. Caffrey, Perper, Tomashefski, and Crouch did not address the issue of total disability.

I previously found Dr. Bush's and Spagnolo's opinions to be the best reasoned and that Drs. Naeye and Castle's opinions were entitled to less weight. Accordingly, I conclude that the miner was totally disabled from a respiratory standpoint by the medical opinion evidence.

Weighing all of the evidence together, in light of the miner's former job description and duties, the qualifying blood gas studies and the more thorough medical opinion of record discussing the miner's impairment in relation to the work performed, I find that Claimant has established total disability from performing his usual coal mine work.

Change in Condition

As Claimant demonstrated he is totally disabled, an element of entitlement previously adjudicated against him, I find that he has demonstrated a material change in condition pursuant to 20 C.F.R. § 725.309 and § 725.310. After evaluating all of the record evidence, including all of the x-ray, CT scan and medical opinion reports, I find that the miner has still not demonstrated the existence of pneumoconiosis.

Disability Causation

A miner shall be considered totally disabled due to pneumoconiosis if his total disability is due, at least in part, to pneumoconiosis. The Fourth Circuit Court of Appeals held that pneumoconiosis must be a "contributing cause" to the miner's disability. *Hobbs v. Clinchfield Coal Co.*, 917 F.2d 790, 792 (4th Cir. 1992); *Robinson v. Pickands Mather & Co.*, 914 F.2d 35, 38 (4th Cir. 1990). As the miner was unable to establish the existence of pneumoconiosis, he is therefore unable to show that pneumoconiosis was a "contributing cause" of his disability.

Entitlement to Benefits

Upon consideration of all of the evidence of record, old and new, I find that the Claimant has failed to show that the miner was totally disabled due to pneumoconiosis during his lifetime. Therefore, the miner's claim must be denied.

## Survivor's Claim

### Length of Coal Mine Employment

The parties have stipulated and I find that the miner was a coal miner within the meaning of the Act for at least 23 years. Tr. 6.

### Date of Filing

The parties have stipulated and I find that Claimant filed her claim for benefits under the Act on January 29, 2001. Tr. 8.

### Responsible Operator

The parties have stipulated and I find that Elkay Mining Company is the responsible operator and will provide payment of any benefits awarded to Claimant. Tr. 6.

### Eligible Survivor

Claimant has not remarried and is eligible for benefits under the Act as she is the surviving spouse of the miner. Tr. 7, 9.

### Standard of Review

The administrative law judge need not accept the opinion of any particular medical witness or expert, but must weigh all the evidence to draw conclusions and inferences. *Lafferty v. Cannelton Industries, Inc.*, 12 B.L.R. 1-190 (1989); *Stark v. Director, OWCP*, 9 B.L.R. 1-36 (1986); *Todd Shipyards Corp. v. Donovan*, 300 F.2d 741 (5th Cir. 1962). The adjudicator's function is to resolve the conflicts in the medical evidence; those findings will not be disturbed on appeal if supported by substantial evidence. *Lafferty,* 12 B.L.R. 1-190; *Fagg v. Amax Coal Co.*, 12 B.L.R. 1-77 (1988), *aff'd*, 865 F.2d 916 (7th Cir. 1989); *Short v. Westmoreland Coal Co.*, 10 B.L.R. 1-127 (1987); *Piccin v. Director, OWCP*, 6 B.L.R. 1-616 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983).

In considering the medical evidence of record, an administrative law judge must not selectively analyze the evidence. *See Wright v. Director, OWCP*, 7 B.L.R. 1-475 (1984); *Hess v. Clinchfield Coal Co.*, 7 B.L.R. 1-295 (1984); *Crider v. Dean Jones Coal Co.*, 6 B.L.R. 1-606 (1983); *Peabody Coal Co. v. Lowis*, 708 F.2d 266, 5 B.L.R. 2-84 (7th Cir. 1983); *see also Stevenson v. Windsor Power House Coal Co.*, 6 B.L.R. 1-1315 (1984). However, the weight of the evidence, and determinations concerning credibility of medical experts and witnesses, is for the administrative law judge. *Mabe v. Bishop Coal Co.*, 9 B.L.R. 1-67 (1986); *Brown v.*

*Director, OWCP*, 7 B.L.R. 1-730 (1985); *see also Roberts v. Bethlehem Mines Corp.*, 8 B.L.R. 1-211 (1985); *Henning v. Peabody Coal Co.*, 7 B.L.R. 1-753 (1985); *Peabody Coal Co. v. Benefits Review Board*, 560 F.2d 797, 1 B.L.R. 2-133 (7th Cir. 1977).

As the trier-of fact, the administrative law judge has broad discretion to assess the evidence of record and determine whether a party has met its burden of proof. *Kuchwara v. Director, OWCP*, 7 B.L.R. 1-167 (1984). In considering the evidence on any particular issue, the administrative law judge must be cognizant of which party bears the burden of proof. Claimant has the general burden of establishing entitlement and the initial burden of going forward with the evidence. *See White v. Director, OWCP*, 6 B.L.R. 1-368 (1983).

Entitlement: In General

To establish entitlement to survivor's benefits, a claimant must establish that the miner had pneumoconiosis, that the miner's pneumoconiosis arose out of coal mine employment, and that the miner's death was due to pneumoconiosis. 20 C.F.R. §§ 718.3, 781.202, 718.203, 718.205(a); *Haduck v. Director, OWCP*, 14 B.L.R. 1-29 (1990); *Neeley v. Director*, OWCP, 11 B.L.R. 1-85 (1988); *Boyd v. Director, OWCP*, 11 B.L.R. 1-39 (1988). For survivor's claims filed on or after January 1, 1982, the miner's death will be considered due to pneumoconiosis if pneumoconiosis was the cause of the miner's death, if pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, if death was caused by complications of pneumoconiosis, or if the presumption related to complicated pneumoconiosis, set forth at § 718.304, is applicable. 20 C.F.R. § 718.205(c)(1)–(3). Pneumoconiosis is a substantially contributing cause of death if it hastened the miner's death. 20 C.F.R. § 718.205(c )(5); *Shuff v. Cedar Coal Co.*, 967 F.2d 977, 16 B.L.R. 2-90 (4th Cir. 1992).

The Existence of Pneumoconiosis

Thirty U.S.C. § 902(b) and 20 C.F.R. § 718.201 define pneumoconiosis as a "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." The definition is not confined to "coal workers' pneumoconiosis," but also includes other diseases arising out of coal mine employment, such as anthracosilicosis, anthracosis, massive pulmonary fibrosis, progressive massive fibrosis, silicosis, or silicotuberculosis. 20 C.F.R. § 718.201. The term "arising out of coal mine employment" is defined as including "any chronic pulmonary disease resulting in respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."

The Claimant has the burden of proving the existence of pneumoconiosis. The Regulations provide that the existence of pneumoconiosis can be established by (1) a chest x-ray meeting the criteria set forth in § 718.202(a); (2) a biopsy or autopsy conducted and reported in compliance with § 718.106; (3) application of the irrefutable presumption for "complicated pneumoconiosis" found in § 718.304; or (4) a determination of the existence of pneumoconiosis

made by a physician exercising sound judgment, based upon certain clinical data and medical and work histories, and supported by a reasoned medical opinion. 20 C.F.R. § 718.202(a).

In *Island Creek Coal Co. v. Compton*, 211 F.3d 203 (4th Cir. 2000), the United States Court of Appeals for the Fourth Circuit held that the administrative law judge must weigh all evidence together to determine whether the miner suffered from coal workers' pneumoconiosis.

In the living miner's claim, I found that the miner did not establish the existence of pneumoconiosis by a preponderance of the chest x-ray, CT scan, biopsy and autopsy evidence, pursuant to 20 C.F.R. § 718.202(a). Nor did I find that the miner was eligible for the enumerated presumptions set forth in 20 C.F.R. §§ 718.304, 718.305, and 718.306. As the above evidence set forth in the living miner's claim is the exact same evidence that is required to make the determination of pneumoconiosis in the survivor's claim and no additional evidence was submitted in connection with the survivor's claim, I hereby incorporate my findings in the miner's claim into the survivor's claim. Accordingly, I find that the Claimant has not met her burden of establishing the existence of pneumoconiosis by a preponderance of the objective medical evidence. In addition, I note the Claimant is still not eligible for the presumptions set forth in 20 C.F.R §§ 718.304, 718.305, and 718.306.

Claimant may also prove the existence of pneumoconiosis by a determination of the existence of pneumoconiosis made by autopsy report or a physician exercising sound judgment, based upon certain clinical data and medical and work histories, and supported by a reasoned medical opinion. 20 C.F.R. § 718.202(a).

As a preliminary matter, I note that the autopsy prosector, the radiologists, and all of the physicians rendering opinions in this matter agreed that the miner suffered from a pulmonary fibrosis. In addition, all of these physicians noted the presence of small amounts of black pigment in the lung. The amended Regulations expand the definition of pneumoconiosis to include anthracosis, and massive and progressive pulmonary fibrosis. 20 C.F.R. § 718.201. In addition, in *Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpublished), the Benefits Review Board held that anthracosis found in the lymph nodes may be sufficient to establish the existence of pneumoconiosis. Accordingly, I note that the autopsy prosector, and Drs. Caffrey, Perper, Bush, Tomashefski, and Crouch all noted the presence of anthracotic pigment in the lymph nodes. Dr. Naeye stated that he does not use changes in the lymph nodes to diagnose coal workers' pneumoconiosis, and Drs. Spagnolo, Castle, and Jarboe did not address the issue.

However, it is not enough that the pigment and fibrosis are present. There must also be evidence that the fibrosis is a result of a reaction to the coal deposits. *See Hapney v. Peabody Coal Co.*, 22 B.L.R. 1-106 (2001). Of the physicians rendering opinions, all of them but Dr. Perper concluded that the miner suffered from idiopathic pulmonary fibrosis that was unrelated to coal dust exposure and that the amount of dust in the miner's lungs was too minimal to cause the extent of reaction seen in the miner's lung tissue.

As I noted in the living miner's claim, Dr. Perper's diagnosis of "severe coal workers' pneumoconiosis of the interstitial pulmonary fibrosis type" is unclear without further explanation

and I am unable to draw the necessary inferences to give weight to this diagnosis. In addition, I found that Dr. Perper's other diagnoses were not well supported by the objective evidence in the record. Specifically, I noted that his finding of severely abnormal ventilatory studies directly contradicts the evidence of record. Finally, I noted that Dr. Perper based his opinion on the miner's "more than forty years exposure to coal mine dust" without observing that the miner worked on the surface and without discussing the miner's dust exposure. Therefore, I found then, as I find now, that Dr. Perper's opinion is not well-reasoned and is entitled to little weight.

Of the other pathologists rendering decisions as to the existence of pneumoconiosis, I found that the reports of Drs. Bush and Tomashefski were the best documented and best reasoned opinions. In addition, I found that Dr. Spagnolo's opinion was also entitled to great weight. In particular, I find that Drs. Bush and Spagnolo best explained how they reached their diagnoses of idiopathic pulmonary fibrosis, as opposed to coal dust exposure, in relation to all of the objective medical evidence, including the miner's physical symptoms and the gross physical findings. Accordingly, I find that Claimant has not met her burden of proving the existence of pneumoconiosis by the medical opinion evidence, pursuant to 20 C.F.R. § 718.202(a). In addition, I note that Dr. Perper's opinion alone, without consideration of the contrary physician opinions, is not well-reasoned enough to establish the existence of pneumoconiosis by a preponderance of the evidence.

Finally, pursuant to *Compton*, I find that in weighing all the evidence together under 20 C.F.R. § 718.202(a), Claimant has not met her burden of proving the existence of coal workers' pneumoconiosis. In addition, as Claimant did not establish that the miner suffered from pneumoconiosis, she cannot establish that pneumoconiosis arose from coal mine employment.

Death Due to Pneumoconiosis

The remaining issue is whether the miner's death was due to pneumoconiosis. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that death is deemed "due to pneumoconiosis" if any of the following criteria is met:

1) competent medical evidence established that the miner's death was due to pneumoconiosis; or
2) pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or the death was caused by complications of pneumoconiosis; or
3) the presumption of § 718.304 [complicated pneumoconiosis] is applicable.

Pursuant to § 718.205(c)(5), pneumoconiosis is a substantially contributing cause of a miner's death if it hastens the miner's death.

There is no evidence that pneumoconiosis was the direct cause of the miner's death; therefore, Claimant has not proven death due to pneumoconiosis pursuant to § 718.205(c)(1). There is no evidence the miner suffered from complicated pneumoconiosis and therefore Claimant has not established death due to pneumoconiosis pursuant to § 718.205(c)(3).

I found that the preponderance of the medical evidence pursuant to 20 C.F.R. § 718.202(a) did not establish the existence of pneumoconiosis. Therefore, as the Claimant was unable to establish that the miner suffered from pneumoconiosis, she cannot show that the miner's death was due to pneumoconiosis pursuant to § 718.205(c).

## Entitlement to Benefits

As Claimant has failed to establish all of the requisite elements of entitlement, I find that she is not entitled to benefits under the Act.

## Attorney's Fees

The award of attorney's fees under the Act is permitted only in cases in which the claimant is found to be entitled to the receipt of benefits. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to these Claimants for the representation services rendered to them in pursuit of the claims.

## ORDER

The claims of Thomas A. Jones (deceased) and Frances Jones, survivor of Thomas A. Jones, for black lung benefits under the Act are hereby DENIED.

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MICHAEL P. LESNIAK
Administrative Law Judge

NOTICE OF APPEAL RIGHTS. Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this Decision and Order was filed in the Office of the District Director, by filing a notice of appeal with the Benefits Review Board at P.O. Box 37601, Washington, DC 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esq., Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2117, 200 Constitution